出國報告（出國類別：開會）

2025 年日本亞太風濕病學會聯盟醫學會議心得報告

服務機關：高雄榮民總醫院兒童醫學部
姓名職稱：翁根本/部主任
派赴國家：日本
出國期間：2025/09/02-2025/09/08
報告日期：2025/09/25

摘要

2025 年亞太風濕病學會聯盟醫學會議於日本福岡舉行，時間是 2025 年 9 月 2 日至 2025 年 9 月 8 日，本人有幸參與此會議，發表論文『血漿 S100A12 有助於川崎症診斷和促進中性白血球與血管內膜細胞的黏著』，收穫良多。上千多位來自世界各國的醫師專家來參與這個盛會，會議內容是探討各種風濕免疫疾病、血管炎和川崎症相關免疫疾病問題，非常多樣豐富，且有機會和其他國家專家交流合作，個人有吸收到相當多新知，對於照顧川崎氏症病童有相當多的幫助。本人和其他各國學者有深入討論，收穫很多，這些交流經驗有助於資料整理和論文發表。參加會議的每一天，都有滿滿收穫和豐富資料，依依不捨離開福岡，希望很快就能再和這些學者做學術經驗上的交流。

關鍵字：川崎氏症、S100A12、中性白血球的黏著

目次

一、目的

2025 年亞太風濕病學會聯盟醫學會議於日本福岡舉行，時間是 2025 年 9 月 3 日至 2025 年 9 月 7 日，本人參與此會議，並發表論文『血漿 S100A12 有助於川崎症診斷和促進中性白血球與血管內膜細胞的黏著』。

二、過程

2025-9-2

由高雄搭機至日本福岡。

2025-9-3

到會場報到，並參加會議，今天是超音波專題討論會。

2025-9-4

今天會議主題是風濕性疾病、免疫學、IL-6 發炎蛋白質探討等題目，做深入教學和討論，其中 IL-6 發炎蛋白質探討專題討論會，對我的臨床照顧和研究幫助很大。

2025-9-5

今天會議主題是風溼性關節炎標靶藥物治療、紅斑性狼瘡疾病病處置、兒科風溼免疫疾病、血管炎新知、和風濕病基因研究等精彩演講；另一個重頭戲是發表下列論文『血漿 S100A12 有助於川崎症診斷和促進中性白血球與血管內膜細胞的黏著』,有很多學者發表川崎症疾病研究，本人和其他各國學者有深入討論，收穫很多，這些交流經驗有助於資料整理和論文發表。

2025-9-6

今天會議主題是關節炎和紅斑性狼瘡疾病診斷治療新進展，以及幼年型紅斑性狼瘡疾病，後續合併症如肺高壓等，也是和我的研究專業肺高壓治療有關，這方面的新知，對我照顧病人和做相關研究的幫助很大。

2025-9-7

今天會議主題是探討風濕性疾病、紅斑性狼瘡疾病、幼年型風濕性性關節炎、骨關節炎和標靶藥物治療、以及轉譯醫學等，聽完這些演講，收穫很多。

2025-9-8

由日本福岡搭機回國。

三、心得及建議

本人參加這次會議，和其他各國學者有深入討論，收穫很多，這些交流經驗有助於本次投稿論文『血漿 S100A12 有助於川崎症診斷和促進中性白血球與血管內膜細胞的黏著』的資料整理和論文發表。參加會議的每一天，都有滿滿收穫和豐富資料，非常充實。建議大家可以整理自己研究心得，積極參與國際醫學會議，增廣自己見聞，院方也能給予適當獎勵補助。

附錄

CERTIFICATE OF ATTENDANCE

This is to certify that

Professor Kenpen Weng

has attended the
27th Asia-Pacific League of Associations for Rheumatology Congress (APLAR 2025) from Wednesday 3rd September 2025 to Sunday 7th September 2025

Prof Tsutomu Takeuchi
APLAR President

Prof Yoshiya Tanaka
APLAR 2025 Congress Chair

27th Asia-Pacific League of Associations for Rheumatology Congress
Fukuoka, Japan | 3-7 September 2025

Plasma S100A12 Contributes to Kawasaki Disease Prediction and Promotes the Adhesion between Neutrophils and Endothelial cells

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Abstract

Background/Purpose:
Kawasaki disease (KD) could be complicated by severe coronary artery lesion (CAL) without early diagnosis and timely medical interventions. Proteins, nucleotides and metabolites in serum are usually applicable in facilitating disease prediction. The purpose of this study was to demonstrate that serum S100A12 protein facilitated early prediction of KD and was involved in CAL formation by promoting in vitro neutrophil infiltration.

Methods:
To examine whether these biomarkers work in plasma samples, we enrolled 80 fever control (FC) and 80 KD subjects (Table 1) and ELISAs were conducted to measure the concentrations of S100A8, S100A9, S100A12, DEFA1 and ORM1 in plasma samples (Table 2). Further in vitro study of S100A12 was done.

Results:
The five proteins (S100A8, S100A9, S100A12, DEFA1 and ORM1) all reached significant differences and kept levels in KD samples (Fig.1). In other words, their abundance tendencies in plasma samples were consistent with those in serum samples. A prediction model based on these five plasma protein biomarkers reached the auROC, sensitivity and specificity values of 0.967, 0.938 and 0.925, respectively (Fig. 2). In addition, treating human coronary artery endothelial cells (HCAECs) with S100A12 enhanced their adhesion with neutrophils and subsequently promoted in vitro neutrophil infiltration (Fig. 3). Such promotion by working on HCAECs could be attenuated with the administration of specific antibody and intravenous immunoglobulin (IVIG) (Fig. 4).

Conclusion:
Our result demonstrated that evaluating S100A8, S100A9, S100A12, DEFA1 and ORM1 plasma levels may be a good diagnostic tool of KD. Further in vitro study implied that S100A12 could be a potential therapeutic target for KD.

Table 1: Demographic and clinical data of the subjects.

Factors	FC (n=80)	KD (n=80)	p-value
Age (year)	1.69±0.76	1.85±1.56	4.30E-01
Gender (male)	48 (60.00%)	56 (70.00%)	1.90E-01
Platelet (1000/uL)	270.82±119.65	328.40±120.48	2.80E-03
Segment (%)	39.68±17.19	56.28±18.19	1.80E-08
Band (%)	0.69±2.62	0.86±1.91	5.90E-01
Lymphocyte (%)	47.71±16.39	32.73±17.07	6.97E-08
Monocyte (%)	8.27±3.55	5.99±3.37	5.02E-05
Eosinophil (%)	2.15±2.72	3.28±3.37	2.05E-03
Basophil (%)	0.22±0.33	0.22±0.32	1.00E-00
CRP (mg/L)	25.96±28.99	80.30±75.57	1.27E-08
Hemoglobin (g/dL)	11.76±1.07	11.29±0.89	3.08E-03
WBC (1000/uL)	8.70±3.60	12.77±4.87	1.13E-08
GOT (U/L)	46.19±20.59	72.62±105.42	2.92E-02
GPP (U/L)	31.32±29.84	70.03±79.82	7.62E-05

Table 2. The performances of prediction factors.

Prediction factors	auROC	Sen.	Spe.	p-value	95% CI
S100A8	0.837	0.900	0.425	0.002	0.549-0.725
S100A9	0.851	0.750	0.850	0.000	0.791-0.911
S100A12	0.836	0.838	0.738	0.000	0.772-0.900
DEFA1	0.883	0.925	0.712	0.000	0.830-0.937
ORM1	0.879	0.788	0.887	0.000	0.825-0.933
Combination	0.967	0.938	0.925	0.000	0.938-0.997

Figures

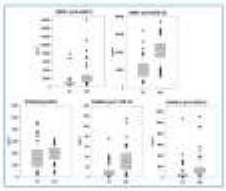


Figure 1. The results of ELISA on plasma samples.

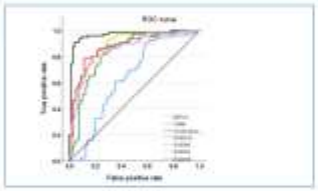


Figure 2. The performances of the factors on predicting KD.

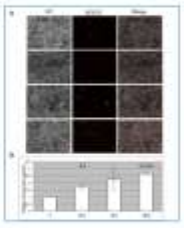


Figure 3. The results of adhesion assays with HCAECs treated with or without S100A12 protein.

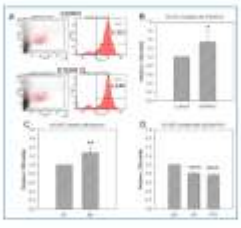
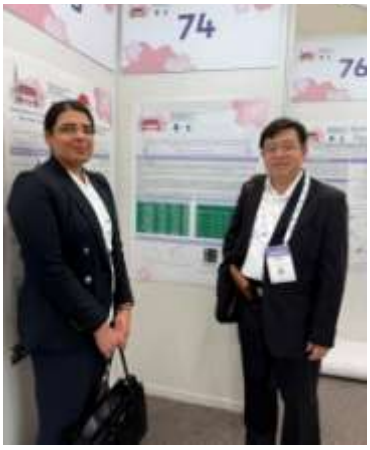


Figure 4. The results of LTEM assays with HCAECs manipulated.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Poster Gallery A
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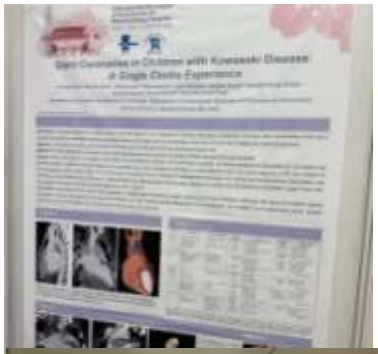

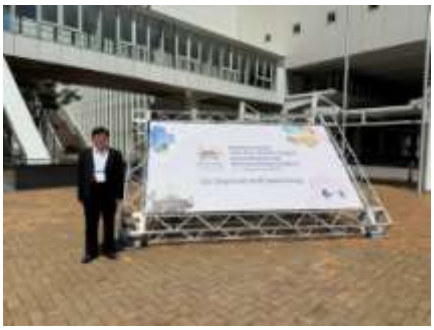


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